Amendments to the Specification:

Please replace the paragraph beginning on page 24, line 3 with the following amended paragraph:

Fig 4A-B shows the assessment of C4 activation with purified IgG (A) and IgM antibodies (B), respectively, in concentrations as indicated, which were incubated on mannan coated plates with MBL-deficient serum in the absence or presence of mAb 2204 anti-C1q.

Please replace the paragraph beginning on page 24, line 3 with the following amended paragraph:

Fig 5A-H shows complement activation *via* the LP and the CP; Complement activation was induced by incubation of different concentrations of NHS on plates coated with IgM for CP activation ~~(A)~~(A-D) or with mannan for LP activation ~~(B)~~(E-H), in the presence or absence of mAb 2004 (20 μg/ml). Activation and binding of complement was demonstrated by detection of C1q (A and E), C4 (B and F), C3 (C and G), and C5b-9 (D and H) using specific mAb.

Please replace the paragraph beginning on page 24, line 3 with the following amended paragraph:

Fig 6A-B shows activation of the alternative pathway; NHS was incubated on plates coated with mannan, LPS, or BSA, in a calcium-free buffer (GVB/MgEGTA) to block activation of the CP and the LP. Binding of C3 (A) and C4 (B), respectively, was subsequently assessed.

Please replace the paragraph beginning on page 29, line 33 with the following amended paragraph:

The C4 depletion, as measured by OD415 in ELISA, in the absence or presence of mAb 2204 anti-C1q is shown in Fig 4. Pre-incubation of mannan-coated plates with purified IgG (Fig 4A) or IgM (Fig 4B) induced a dose-dependent deposition of C4 on mannan upon addition of MBL-deficient serum (*BB* genotype), whereas no complement activation could be detected with this serum alone. Activation of C4 induced by anti-mannan Ab was completely inhibited by addition of a C1q-inhibitory Ab in the MBL-deficient serum, clearly indicating that mannan-binding IgG and IgM can restore complement activation by mannan in MBL-deficient serum by the activation of the classical pathway of complement, in the absence of functional MBL.

Please replace the paragraph beginning on page 30, line 27 with the following amended paragraph:

The complement activation cascade was further studied by using mAb to detect binding of specific complement components upon their activation *via* the CP and the LP, respectively. Incubation of NHS on immobilized IgM resulted in a dose-dependent deposition of C1q, C4, C3, and C5b-9 to the plate ~~(Fig 5A)~~ (Figs. 5A-D). Binding the C1q and a subsequent complement activation induced by IgM could be completely inhibited by mAb 2204. Incubation of NHS on immobilized mannan resulted in a dose-dependent binding of C4, C3 and C5b-9, whereas binding of C1q was hardly detectable ~~(Fig 5B)~~ (Figs. 5E-H). Complement activation by mannan was only slightly inhibited by the addition of mAb 2004. Therefore, addition of mAb 2004 to serum allows the specific detection of LP activation by using mannan as ligand without any interference of the CP.

Please replace the paragraph beginning on page 31, line 24 with the following amended paragraph:

In order to enable the detection of all complement activation pathways in one assay system, the activation of the alternative pathway in an ELISA system was also studied. In contrast to the LP and the CP, activation of the AP is calcium-independent. Therefore, a calcium-free buffer was used, thus excluding involvement of the CP and the LP. As previously described (Fredrikson G.N., *et al., J. Immunol. Methods* 166, 263-270, 1993), incubation of NHS in a buffer containing EGTA and Mg** on plates coated with LPS resulted in a does-dependent deposition of C3 (Fig 6). Some activation of C3 was also observed on plates coated with BSA only, most likely due to spontaneous activation of the AP. Surprisingly, a strong activation of C3 was also observed when NHS was incubated on mannan-coated plates by using the same conditions, suggesting that a mannan may also support the activation of the AP. The detection of C3 was reduced until background levels when EDTA was present in the complement source (not shown). As expected from an AP-dependent mechanism, C3 activation in calcium-free buffers required a serum concentration that is about 10-fold higher than that required for C3 activation by mannan in a calcium-containing buffer *via* the LP (compare Fig 6 with Fig 5B). Although C3 activation was clearly detectable in a calcium-free buffer (Fig 6A), no activation of C4 could be established ~~(Fig 6)~~, suggesting that under these conditions activation of C3 (Fig 6B) is independent of MBL binding and C4 activation.